REMARKS

Claims 6, 13, 16, 19-21, 26, 29, 30, 41, 45, 48, 50, 65, 68, and 70 are pending. By this Amendment, claim 6 is canceled, claims 13, 16, 19, 20, 26, 29, 41, 45, 48, and 50 are amended, and new claim 72 is added. After entry of this Amendment, claims 13, 16, 19-21, 26, 29, 30, 41, 45, 48, 50, 65, 68, 70, and 72 will be pending.

Claim 6 was rejected under 35 U.S.C. § 101 because the claimed recitation of a use, without setting forth any steps involved in the process, results in an improper definition of a process. Claim 6 has been canceled without prejudice.

Claims 6, 13, 16, 19-21, 26, 30, 41, 45, 48, 50, 65, 68, and 70 were rejected under 35 U.S.C. § 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. Claim 6 has been canceled without prejudice.

Claim 13 has been amended to claim a method for potentiating normal or deficient insulin function on glucose metabolism in a mammal when measured on an intravenous glucose tolerance test. Claim 13 further claims the step of administering an effective amount of one or more compounds.

Claim 16 has been amended to depend from claim 13 and as such includes all the limitations thereof. Hence, the method of claim 16 is accomplished by administering to a mammal one or more compounds as defined in claim 13 in a quantity that is about 4% to about 60% of said mammal's diet.

Claims 19 and 29 have been amended to correct the lack of antecedent basis for "hyperglycemia."

Claims 20, 48, and 50 have been amended to claim the fish protein as "cod protein."

Claim 45 has been amended to depend from claim 29 and to claim "said hyperglycemia is the result of Type 1 or Type 2 diabetes."

Claim 26 has been amended to depend from claim 13 and to overcome the various objections under 35 U.S.C. § 112, second paragraph.

It is respectfully submitted that amended claims 13, 16, 19, 20, 26, 29, 41, 45, 48, and 50 are allowable. Claims 21, 65, 68, and 70 variously depend from these claims and are allowable at least for the reasons above.

Claims 26, 29, and 30 were rejected under 35 U.S.C. § 102(e) as being anticipated by U.S. Patent No. 6,143,786; and under 35 U.S.C. § 102(b) as being anticipated by U.S. Patent Nos. 5,843,886 and 5,830,434.

Claim 26 has been amended to depend from claim 13 and is therefore believed to be in condition for allowance. Claims 29 and 30 depend from claim 26 and are therefore allowable at least for these reasons.

Claims 6, 13, 16, 19-21, 26, 29, 30, 41, 45, 48, 50, 65, 68, and 70 were rejected under 35 U.S.C. § 103(a) as being unpatentable over U.S. Patent No. 4,584,197 to Takasaki et al. Claims 6, 13, 16, 19-21, 26, 29, 30, 41, 45, 48, 50, 65, 68, and 70 were rejected under 35 U.S.C. § 103(a) as being unpatentable over Gohman, Weiner or Taylor in view of Takasaki.

Claim 6 has been canceled without prejudice.

Claim 13 has been amended to claim a method "for potentiating normal or deficient insulin function on glucose metabolism in a mammal when measured on an intravenous glucose tolerance test." Support for the term "intravenous glucose tolerance test" can be found on page 4, line 20, of the application as filed. No new matter has been added.

The cited references neither teach nor suggest the method of amended claim 13. Takasaki et al. claim that their extracts exert "insulin-like function," whereas the Applicants have observed that fish protein extracts potentiate or enhance insulin action on glucose metabolism. Applicants' data do not show any insulin-like function of fish proteins (or fish protein-derived amino acids) on glucose metabolism. Rather, the Applicants have found that the addition of fish protein to a high-fat diet prevented the development of obesity-linked insulin resistance by enhancing the action of insulin in skeletal muscle. This noted improvement of insulin action on glucose metabolism was confirmed in vitro using cultured myocytes incubated with a mixture of amino acids found in the plasma of rats treated with fish protein extracts. For these reasons, it is believed that there is nothing in Takasaki nor remaining cited references that teaches or suggests the invention as claimed in amended claim 13.

Therefore, claim 13 is allowable. Claims 16, 19, 20, 21, 26, 29, 30, 41, 45, 48, 50, 65, 68, and 70 ultimately depend from claim 13 and are allowable at least for these reasons.

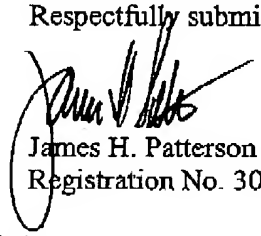
Applicant has added a new claim 72. Support for new claim 72 can be found on page 1, lines 21-23, of the application as filed: "Although most studies have examined the role of high-fat [3-7], low-soluble fiber [8] or high-sucrose diets [9] in the development of an impaired insulin action," as well as at page 5-6, lines 25-3: "Consequently, a study was devised to test the effects of dietary cod and soy proteins compared with casein on peripheral insulin sensitivity of rats made obese by feeding a high-fat/sucrose diet. The high-fat/sucrose fed rat is a well-established animal model of insulin resistance reproducing the common form of the abdominal (visceral) obese insulin-resistant syndrome seen in humans [25-29]." No new matter has been added.

Claim 72 depends from claim 13 and is allowable at least for the reasons set forth above.

In view of the foregoing, it is submitted that this application is in condition for allowance. Favorable consideration and prompt allowance of the application are respectfully requested.

The Examiner is invited to telephone the undersigned if the Examiner believes it would be useful to advance prosecution.

Respectfully submitted,



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